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ABSTRACT

Senescence patterns are highly variable across the animal kingdom. However, while empirical evidence of actuarial senescence in vertebrates is accumulating in the wild and life history correlates of actuarial senescence are increasingly identified, both the extent and variation of reproductive senescence across species remain poorly studied. Here, we performed the first large-scale analysis of female reproductive senescence across 101 mammalian species that encompassed a wide range of Orders. We found evidence of reproductive senescence in 68.31% of the species, which demonstrates that reproductive senescence is pervasive in mammals. As expected from allometric rules, the onset of reproductive senescence occurs later and the rate of reproductive senescence decreases with increasing body mass and delayed age at first reproduction. Moreover, for a given pace of life, females displaying a high level of multiple mating and/or with induced ovulation senesce earlier than females displaying a low level of multiple mating and/or with spontaneous ovulation. These results suggest that both female mating behavior and reproductive physiology shape the diversity of reproductive senescence patterns across mammals. We propose future avenues of research regarding the role played by environmental conditions or reproductive features (e.g. type of placentation) on the evolution of reproductive senescence.

Keywords: Aging, Comparative biology, Fertility, Life-history, Reproduction

1. Introduction

From an evolutionary biology perspective, the process of senescence corresponds to a decrease in the age-specific contribution to fitness (Cohen et al., 2020; Gaillard and Lemaître, 2020; Monaghan et al., 2008). Therefore, senescence is mostly studied through a decline in demographic rates with increasing age (i.e. actuarial and reproductive senescence). Historically, senescence was thought to not show up in the wild because animals were expected to die from harsh environmental conditions or anthropogenic effects (e.g. hunting) before experiencing a decline in age-specific survival or reproductive rates (Nussey et al., 2013). Thanks to the increasing number of long-term field studies - where individuals are monitored from birth to death (Clutton-Brock & Sheldon 2010), and the subsequent demographic analyses of these populations, the view that demographic senescence does not occur in the wild has been totally revisited (Nussey et al. 2013). Yet, after pioneer descriptive studies suggesting that mortality increases in late life (e.g. Caughley, 1966), empirical evidence currently indicates that actuarial senescence is pervasive across vertebrates with determinate growth in the wild (Brunet-Rossinni and Austad, 2006; Nussey et al., 2013), whereas reproductive senescence was considered to be inexistent in the wild for a much longer period (e.g. Caughley, 1976). On the contrary, the negative effects of age on reproductive performance have been documented in laboratory rodents (e.g. Leslie and Ranson, 1940) and human populations (e.g. Tietze, 1957) for a while. The first reports of reproductive senescence in the wild only emerged in the late 70s (Coulson and Horobin, 1976; Ollason and Dunnet, 1978; Perrins and Moss, 1974; Sinclair, 1977), following the insightful work by Emlen (1970), who stated that age-specific reproductive rates should increase to a peak, then stay relatively constant during a prime-age stage (i.e. the period of adulthood between the beginning of the reproductive peak and the onset of reproductive senescence) before declining in late life.

Empirical evidence of reproductive senescence from wild populations of vertebrates is now compelling (Nussey et al., 2013). So far, these case studies have been almost entirely performed on females (but see Lemaître and Gaillard, 2017 for a review of evidence in males), simply because accurate paternity assignments in the wild require the use of molecular tools. Age-specific declines in a wide array of traits reflecting female reproductive success have been documented, such as breeding proportions (e.g. Photopoulou et al., 2017), birth rates (e.g. Lee et al., 2016), number of offspring produced at birth (e.g. Sparkman et al., 2017) or at the end of parental care (i.e. at weaning or fledgling, e.g. Thorley et al., 2020), and

offspring survival (e.g. Karniski et al., 2018). Interestingly, it has been suggested that female reproductive senescence patterns can markedly differ across species (Baudisch and Stott, 2019; Jones et al., 2014), even between closely related species. For instance, a population-level comparison of reproductive senescence in terms of breeding success across three albatross species has revealed that the age at the onset of reproductive senescence was much earlier in the wandering albatross (*Diomedea exulans*) than in both the black-browed albatross (*Thalassarche melanophris*) and the grey-headed albatross (*Thalassarche chrysostoma*) (Froy et al., 2017). Likewise, the southern fulmar (*Fulmarus glacialoides*) showed clear evidence of senescence in breeding success, whereas the snow petrel (*Pagodroma nivea*) did not (Berman et al., 2009). Despite these reports of contrasted patterns of age-specific decline in reproductive performance with increasing age, there has been so far no attempt to quantify the occurrence and the variation in reproductive senescence across a wide range of species, or to identify the ecological and biological factors underlying the diversity of patterns. This contrasts with the increasing number of comparative analyses that focused on actuarial senescence (Bronikowski et al., 2011; Lemaître et al., 2020c; Péron et al., 2019b; Ricklefs, 1998, 2006; Ricklefs and Scheuerlein, 2001), which have notably revealed that life-history strategies (e.g. Garratt et al., 2013; Ricklefs, 2010) and environmental conditions (Colchero et al., 2019; Lemaître et al., 2013; Tidière et al., 2016) modulate both the onset and the rate of actuarial senescence. So far, most comparative analyses of age-specific reproductive data in mammals have focused on the evolution of post-reproductive lifespan (e.g. Alberts et al., 2013; Cohen, 2004; Ellis et al., 2018) and did not investigate among-species differences in the onset or rate of reproductive senescence. Only Jones and colleagues (2008) analyzed interspecific differences in female reproductive senescence patterns (i.e. measured as the age-specific decline in the number of recruited offspring) across 19 species of birds and mammals. They found that the rate of reproductive senescence decreased with increasing generation time, a reliable measure of the species position along the slow-fast continuum (Gaillard et al., 2005). Thus, slow-living species had a lower rate of reproductive senescence than fast-living species (Jones et al., 2008; see also Gaillard et al., 2016).

The aim of this work is twofold. First, we assess whether female reproductive senescence is the rule rather than the exception across mammals by accurately quantifying reproductive senescence using a large sample of mammalian species that display a high diversity of lifestyles and life history strategies. This first step allowed quantifying among-species variation in both the onset and the rate of reproductive senescence. Second, we investigate the role of several ecological, biological and life history traits in shaping the

diversity of reproductive senescence patterns observed across mammalian females. Among these factors, we focus on the role played by the phylogeny, the species position along the slow-fast continuum, the mating behavior and the ovulation mode.

Following comparative analyses of many life history traits (e.g. Kamilar and Cooper, 2013; Wootton, 1987), notably the rate of actuarial senescence (e.g. Lemaître et al., 2020b), we expected closely related species to share more similar reproductive senescence patterns than distant species across the phylogeny. We also expected the onset and the rate of senescence to occur earlier and to increase, respectively, with increasingly fast-living life history as a direct consequence of the covariation among all biological times (i.e. life history traits expressed in time units that positively covary across species, see Gaillard et al., 2016; Ronget and Gaillard, 2020). Moreover, we investigated whether the number of mating partners and the ovulation mode influenced the timing and intensity of reproductive senescence. In most mammals, females generally copulate with more than one male during a given reproductive event (Gomendio et al., 1998; Hayssen and Orr, 2017; Soulsbury, 2010). The propensity of females to mate repeatedly within a reproductive event increases from monogamous species to polyandrous or polygynandrous species, which is considered as a way to increase fitness through genetic benefits (e.g. increased genetic diversity among offspring, Jennions and Petrie, 2000; Stockley, 2003). However, multiple mating promotes sperm competition (i.e. when sperm from two or more males compete to fertilize a given set of ova, Parker, 1970), which in turn can lead to intense sexual conflicts between sexes (Stockley, 1997). Physiological and subsequent fertility costs associated to these sexual conflicts have been widely documented (see Arnqvist and Rowe, 2005; Stockley, 1997 for reviews) and might increase with female age. Thus, the risk of contracting infectious diseases increases steadily with age in species displaying high levels of multiple mating (Nunn et al., 2014) and the negative consequences of such infections in terms of female fertility are likely to be amplified at old ages due to the progressive (and taxonomically widespread) decline un immune performance throughout the lifetime (Peters et al., 2019). We thus tested whether the degree of multiple mating by females leads to an earlier and/or steeper reproductive senescence. We also investigated whether the ovulation mode influences mammalian reproductive senescence. Ovulation is a physiological process governed by complex interactions between hormonal levels and external cues (e.g. photoperiod, mating) (Hayssen and Orr, 2017). Mammalian females are typically divided between spontaneous and induced ovulators (Soulsbury and Iossa, 2010). When spontaneous, ovulation is mostly triggered by endogenous hormonal changes, whereas with induced ovulation, the mating event triggers a

physiological cascade that leads to ovulation. Comparative analyses performed so far have revealed that the intensity of sperm competition is stronger in species with spontaneous ovulation (Iossa et al., 2008; Soulsbury and Iossa, 2010). Since sperm competition can be associated with long-term aging costs in females, including a decline in age-specific physiological performance and fertility in females (Lemaître et al., 2020a; Stockley, 1997), we expect females from species with spontaneous ovulation to suffer from an earlier and/or steeper reproductive senescence than females from species with induced ovulation.

2. Material and methods

2.1. Data collection

Age-specific reproductive data for females were extracted from an unpublished database (entitled 'Malddaba') built and managed by VR, JFL and JMG, and currently under development. This database contains sex- and age-specific demographic data for wild populations of mammals gathered from published life tables or extracted from graphs (using WebPlotDigitizer (https://automeris.io/WebPlotDigitizer/)) over the past few years. We focused mainly on reporting the m_x series, defined as number of daughters alive at birth that are produced by a female of age x. For each study we collected the mean m_x value at each age, as well as corresponding sample size. In some studies, the sample size was not directly reported but the information on the age distribution of females was provided. In those cases, we extracted the age distribution and used the number of females expected to be alive at age x as the sample size of the m_x series. When m_x series were not available, we reported either the age-specific litter size or the age-specific pregnancy (or birth) rates with the sample sizes (i.e. in monotocous species, the m_x is equivalent to the birth rates divided by 2 when assuming a balanced sex ratio at birth). Only one population per species was considered in this study. When age-specific reproductive parameters were available for several populations of the same species, we selected the one based on the best data quality and with the largest sample size (see *Supplementary datasets* for the full list of populations included in our comparative analysis).

Based on the accuracy of the methods used to assign the age of the individuals, we distinguished two main categories of studies. The first type of study (i.e. longitudinal data) corresponds to age-specific reproductive estimates obtained from the long-term monitoring of individuals marked at birth or early in life when age can be accurately identified. In the second type of studies (i.e. cross-sectional data), age is generally estimated through indirect

methods (e.g. tooth wear) and subject to uncertainty. These cross-sectional data correspond to age-specific reproductive rates obtained from a single assessment of individual reproductive status using individuals either controlled alive in the population or recovered as dead in the field. In cross-sectional studies performed from dead recoveries, the count of embryos or placental scars often provide estimates of the litter size (e.g. Lieury et al., 2017). Sampled populations were also classified as hunted vs. non-hunted according to the information reported in the original publication. Finally, we used the age-specific reproductive data provided in the life tables to recover the age at first reproduction. We limited our research to wild or semi-captive (i.e. where individuals can freely reproduce) mammalian populations. However, we observed that this selection did not allow including small (and fast) mammalian species, which is easily explained by the lack of long-term field studies for such species. Therefore, to avoid any bias in our interpretation of the diversity of reproductive senescence patterns across mammals, we included in our dataset age-specific reproductive rates gathered in three captive populations of rodents, all belonging to the Cricetidae family (Field vole, *Microtus agrestis*, Orkney vole, *Microtus arvalis* and Tundra vole, *Microtus oeconomus*). At the end of our literature survey, we gathered age-specific reproductive data for 101 mammalian species (including 52 longitudinal studies and 49 cross-sectional studies) encompassing a wide diversity of mammalian Orders (Figure 1). Carnivora and Cetartiodactyla were the most represented Orders (35.6% and 27.7% of the species, respectively) followed by Rodentia and the Primates (16.8% and 11.8% of the species, respectively).

When possible, we extracted data on female body mass from the population where we retrieved age-specific reproductive data. When this information was lacking, we looked for female adult body mass from other populations. In mammals, there is widespread evidence that testes mass (relative to body mass) is a reliable indicator of the level of multiple mating that is experienced by females during a given reproductive event (Harcourt et al., 1995; Lemaître et al., 2009; Ramm et al., 2005; Soulsbury, 2010). Indeed, multiple mating promotes sperm competition among males, which in turn selects for large testes (Parker, 2016). We thus used published reviews to compile data on combined adult testes mass and the corresponding adult male body mass (e.g. Lüpold, 2013). Data on ovulation mode were retrieved from Soulsbury (2010) and Soulsbury and Iossa (2010). All data and associated references are provided in the *Supplementary datasets*.

2.2. Measures of reproductive senescence

In living organisms, age-specific fertility is predicted to "*rise with age to a peak, which may occur at almost any age depending on the sort of organism considered, and then fall*" (Emlen 1970). When looking at age-specific variation in reproductive rates across mammals, this prediction is most likely verified but also associated with substantial variation across species in the strength of the initial increase, the form of the reproductive pattern near the peak of reproduction and the strength of the decrease. For instance, in the bighorn sheep (*Ovis canadensis*), three distinct phases are observed (Figure 2). First, the reproduction rate steadily increases with age from 2 to 4 years of age and then slightly increases during the prime-age stage between 4 and 12 years of age when the reproduction rate peaks and then reproductive senescence begins to occur (i.e. fertility is decreasing with increasing age from 12 years of age onwards). Although many mammalian species display this three-phase pattern, there is still a lot of variation. For instance, the prime-age stage associated with high and constant reproductive rates is not consistently observed. Reproductive rates can slightly but consistently increase (such as in Bighorn sheep, Figure 2) or decrease during this prime-age stage or this plateau phase can simply not exist when reproductive senescence occurs for instance right after the age at first reproduction (e.g. in Short-finned pilot whale, *Globicephala macrorhynchus*, Kasuya and Marsh, 1984). Lastly, reproductive senescence has not been detected in all mammal species studied so far (e.g. Northern elephant seal, *Mirounga angustrirostris*, Le Boeuf and Laws, 1994).

To identify the different phases of age-specific variation in reproductive rates and estimate when possible both the age at the onset of reproductive senescence and the rate of reproductive senescence, we modeled age-specific changes of reproductive rates using segmented models from the R-package *segmented* (Muggeo, 2017). We acknowledge that this approach is not necessarily the most accurate to model age-specific variation in a reproductive trait for each species included in our dataset. However, our goal was to assess at the large scale of mammalian species the existence of reproductive senescence using a standardized method, and when present to estimate both the age at the onset of reproductive senescence and the rate of reproductive senescence. To account for the diversity of age-specific trajectories of reproductive rates, we fitted four models of variation in reproductive rates: a constant model with age, a linear model with age, a model with one threshold age including two linear segments (i.e. one before and one after the threshold age) and a model with 2 threshold ages including 3 linear segments (i.e. one before the first age threshold, one between the two age thresholds, and one after the second age threshold).

Models were fitted by weighting the age-specific reproductive trait by sample size. Standard deviation of the reproductive trait was not used because it was not reported in most cases contrary to sample size. Aggregated mean values per age were used for the model fitting and, as we did not have access to the raw individual data in most cases, we had to make an assumption about the distribution of the mean reproductive trait. Following the central limit theorem, we considered each mean trait value to be normally distributed because individual reproductive traits studied here were either binomial or Poisson distributed. All models were fitted between the age at first reproduction and the last age reported in the study. For three species (Crabeater seal, *Lobodon carcinophagus*; Leopard, *Panthera pardus*; Western Jumping Mouse, *Zapus princeps*) we set the reproductive trait to 0 one year before the age at first reproduction to anchor the models and help reach numeric convergence. For each species, the most parsimonious model was selected using the Akaike Information Criterion (AIC). We calculated AIC weights (AICw) to assess the relative likelihood that a given model was the best among all the three fitted models (Burnham and Anderson, 2002). We selected the model with the lowest AIC. To account for the propensity of AIC to retain a too complex model (Link and Barker, 2006), when the difference in AIC (denoted ΔAIC) of two competing models was less than two units, we retained the simplest model in accordance with parsimony rules, except in two species (Alpine marmot, *Marmota marmota* and Wolf, *Canis lupus*) for which the original studies reported statistical evidence of reproductive senescence using threshold models (i.e. Berger et al., 2015, Stahler et al., 2013). In both cases, we retained the model including senescence and checked that our estimates of the age at the onset of reproductive senescence matched those provided in the original sources. Depending on the model selected, we used a pre-defined procedure to assess the occurrence of reproductive senescence (see *Appendix B* for detailed procedure).

2.3. Statistical analyses of the factors influencing reproductive senescence

To avoid any statistical issue due to phylogenetic inertia (Felsenstein, 1985), all the following analyses involved phylogenetically controlled models. We used Phylogenetic Generalized Least-Squares models (PGLS), with a variance-covariance matrix extracted using the R-package *ape* (Paradis et al., 2004). The strength of the phylogenetic signal on the error structure of each model was assessed with the Pagel's λ (Pagel, 1999, henceforth called 'λ'), which is incorporated into the analysis to control for the phylogenetic dependence among species (Symonds and Blomberg, 2014). Here, λ was estimated with maximum likelihood by using the PGLS command from the R-package *caper* (Orme et al., 2013). In most cases, λ

varies between 0 (no phylogenetic signal) and 1 (the observed pattern is predicted by the phylogeny; similarity among species scales proportionally to their shared evolutionary time following a Brownian motion model; Pagel, 1999). As it is not possible to estimate λ for binary data, we used the *D* statistic for this specific analysis (Fritz and Purvis, 2010), which we estimated with the R-package *caper* (Orme et al., 2013). The *D* statistic is equal to 1 if the trait of interest has a phylogenetically random distribution across the species included in the phylogeny and to 0 if its evolution follows a Brownian motion model (Fritz and Purvis, 2010). For all the phylogenetically-controlled analyses, data were linked to a super-tree of extant mammals that provides information on both topology and branch length (Bininda-Emonds et al., 2008, 2007). To identify factors shaping the interspecific variation in both the onset and the rate of reproductive senescence, our statistical analyses were run in the following four steps.

First, we tested whether the probability to detect reproductive senescence was influenced by the sample size (i.e. total number of reproductive records in the population; log-transformed in the analysis), the hunting status of the population, the type of data (i.e. longitudinal vs. cross-sectional) or the age at first reproduction (to control for the pace of life). For this, we ran phylogenetically controlled logistic regressions with the presence or absence of reproductive senescence using the R-package *phylolm* (Ives and Garland, 2010). To identify the best models of the onset and of the rate of reproductive senescence, we performed a model selection procedure using AIC (see above for a description of the procedure). Second, we estimated the phylogenetic signal in both the onset and the rate of reproductive senescence (see Kamilar and Cooper, 2013 for a similar approach). Third, we focused on all the candidate factors for the whole set of species displaying reproductive senescence ($N = 69$ species). We thus tested whether the onset of reproductive senescence and the rate of reproductive senescence were influenced by both the age at first reproduction and the female body mass (entered as covariates). We also included potential confounding factors such as data quality (entered as a two-level categorical data: longitudinal vs. cross-sectional data) and the hunting status of the population (entered as a two-level categorical data: hunted vs. non-hunted population). As expected, the age at first reproduction was positively associated with the female body mass (slope of 0.20 ± 0.04, $R^2 = 0.31$, $\lambda = 0.83$, on a log-log scale, *Appendix C* - Figure S1). However, we kept both variables in our model selection procedure because the Pearson coefficient of correlation between the age at first reproduction and female body mass ($r = 0.56$) was below the threshold of 0.7 indicating statistical issues due to a collinearity among predictor variables (Dormann et al., 2013). In addition, to get

estimates of the rate of reproductive senescence independently of the onset of reproductive senescence (slope between rate and onset of reproductive senescence of -0.81 ± 0.22, R^2 = 0.17, λ = 0.87, Figure 3), we included the age at the onset of reproductive senescence among our set of covariates in the pool of models seeking to explain interspecific variation in the rate of reproductive senescence. To identify the best models of the onset and of the rate of reproductive senescence, we performed a model selection procedure using the AIC (see above). Fourth, we tested on a subset of species (N = 54) for an effect of relative testes mass on both the onset and the rate of reproductive senescence. For these analyses, we added as covariates both testes mass and male body mass (Freckleton et al., 2002; Gage and Freckleton, 2003) to the factors found to influence reproductive senescence traits in the previous analyses, and we then updated the model selection. Fifth, we analyzed the influence of the ovulation mode (i.e. spontaneous vs. induced ovulation) using another subset of species (N = 40). We followed the exact same procedure by adding the ovulation mode (as a categorical variable) to the previously identified set of factors influencing reproductive senescence and by running model selection, for the onset and the rate of reproductive senescence, respectively. For all the statistical analyses, the onset of reproductive senescence, the rate of reproductive senescence, the age at first reproduction, testes mass and both female and male body mass were log-transformed. All PGLS models were performed with R 4.0.0 (R Core Development Team) using the packages *ape* (Paradis et al., 2004) and *caper* (Orme et al., 2013). Unless otherwise stated, parameter estimates are given as ± one standard error.

3. Results

3.1. Occurrence of female reproductive senescence across mammalian species

Reproductive senescence was detected in 68.31 % of the species (69 out of 101 species). The probability to detect senescence was moderately influenced by the phylogeny (D = 0.61). When looking at the different traits that putatively had an influence on the probability to detect senescence, the selected model contains the data quality, the hunting status and the sample size (*Appendix C*, Table S1; Table 1a). As we could expect, reproductive senescence was more often detected in longitudinal studies (80.77 %, 42 out of 52 studies) than in cross-sectional studies (55.10 %, 27 out of 49 studies) and the probability to detect reproductive senescence increased with the sample size (slope of 0.36 ± 0.15, Table 1a, *Appendix C* - Figure S2). When the sample size was large (> 80 individuals), the probability to detect reproductive senescence was above 0.5 (computed on the entire dataset). Reproductive

senescence was also more often detected in non-hunted populations (73.41 %, 58 out of 79 populations) than in hunted populations (50 %, 11 out of 22 populations).

3.2. Factors modulating female reproductive senescence

The age at the onset of senescence was strongly influenced by the phylogeny (λ=0.96, 95% CI [0.88, 0.99]). Species that displayed a late age at the onset of reproductive senescence were typically long-lived mammals (e.g. Proboscidae), which could confound the effect of the phylogeny (Figure 1). To disentangle these effects, we scaled the age at the onset of reproductive senescence by the age of the oldest individual sampled in the population. The influence of the phylogeny on the standardized age at the onset of reproductive senescence was still statistically significant but lower than the one previously observed for the *absolute* age at the onset of reproductive senescence (λ=0.66, 95% CI [0.22, 0.88]; Figure 1). Three cetaceans known to display extended periods of post-reproductive lifespan (see Péron et al., 2019a) showed very early *relative* ages at the onset of reproductive senescence (i.e. onset located at 11.5%, 24.5% and 35.4% of the lifespan for *G. macrorhynchus*, *O. orca* and *P. crassidens*, respectively). Interestingly, when applying our statistical procedure to study reproductive senescence in human populations (see *Appendix D* for an illustration), which display the longest post-reproductive life ever reported in mammals, we noted a possible association between an early onset of reproductive senescence and the existence of an extended period of post-reproductive lifespan.

The selected model of among-species variation in the age at the onset of reproductive senescence included both age at first reproduction (slope of 0.45 ± 0.12) and body mass (slope of 0.10 ± 0.04) (Table 1b, *Appendix C* - Table S2A). As expected from the existence of both time and allometric constraints on overall life histories, the onset of reproductive senescence increased with both age at first reproduction and female body mass (Figure 4a, 4b). Once scaled, the effect of the age at first reproduction was stronger than the effect of body mass (slope of 0.40 ± 0.11 vs. 0.26 ± 0.12), with an effect 1.54 times stronger. Although not retained in the selected model, data quality and hunting status were also included in the model with the lowest AIC value (*Appendix C* - Table S2A) suggesting that, for a given age at first reproduction and a given body mass, the age at the onset of reproductive senescence tends to be later in populations monitored longitudinally and hunted (Table 1c, *Appendix C* - Figure S3).

We then analyzed the effect of testes mass (relative to body mass) on the onset of reproductive senescence. The selected model only included the age at first reproduction

(*Appendix C* - Table S2B) but the model with the lowest AIC value included an additional negative effect of relative testes mass on the onset of reproductive senescence (slope of -0.12 ± 0.07, Table 1d). For a given age at first reproduction, females with high levels of multiple mating (i.e. species where males carry large testes relative to body mass) tend to show an earlier age at the onset of reproductive senescence (Figure 5). Finally, the onset of reproductive senescence was earlier in species with induced ovulation than in species with spontaneous ovulation (Figure 6). Indeed, the selected model included both the age at first reproduction and the ovulation mode (*Appendix C* - Table S2C, Table 1e). Moreover, the effect of the ovulation mode remained statistically significant when the relative testes mass was added to the model (Table 1e). Interestingly, when considering this model, the effect of testes mass (controlled for body mass) was also statistically significant (Table 1f).

Similar to the onset of reproductive senescence, the rate of senescence was strongly influenced by the phylogeny (λ=0.94, 95% CI [0.84, 0.99], *Appendix C* - Figure S4). The retained model explaining variation in the rate of reproductive senescence across mammalian species included both the age at first reproduction and female body mass (Table S3A), as expected from both time and allometric constraints. Thus, the rate of senescence decreased with increasing age at first reproduction and female body mass (Table 1g, Figure 2c, 2d). However, contrary to what was observed for the age at the onset of reproductive senescence, the effect of body mass on the rate of reproductive senescence was 1.48 times stronger than the effect of the age at first reproduction once both traits were scaled (slope of -0.83 ± 0.22 vs. -0.56 ± 0.20). Here, we can notice that the onset of reproductive senescence was not retained in the selected model (*Appendix C* Table S3A), which highlights that the three biological times included in the analysis positively covaried, with the age at first reproduction being more closely associated to the rate of reproductive senescence than the age at the onset of reproductive senescence. Subsequent analyses did not reveal any detectable influence of either relative testes mass or ovulation mode on the rate of reproductive senescence (*Appendix C* - Table S3B, S3C). Importantly, when we restricted our analyses to species for which age-specific reproductive data are based on m_x series, all results were qualitatively unchanged (*Appendix C* - Table S3D).

4. Discussion

The widespread occurrence of reproductive senescence in mammalian females

Our comparative analysis provides firm evidence that reproductive senescence is widespread across mammals in the wild. More specifically, we found support for an age-specific decline

in reproductive performance in more than two-third (68.3%) of the species included in our analysis, and at least four lines of evidence suggest that we currently underestimate its occurrence.

First, the ability to detect reproductive senescence was much higher when analyzing age-specific reproductive traits from longitudinal trajectories rather than cross-sectional data. A higher accuracy in age assessment of longitudinal data compared to cross-sectional data is most likely to be involved. For instance, while longitudinal studies are performed from known-aged individuals (generally marked near birth or within the early life period), cross-sectional data rely on age estimates most often based on tooth wear, which varies in reliability among species (Hamlin et al., 2000). This difference in the quality of the age estimation thus likely explains our results.

Second, the probability to detect reproductive senescence increased with sample size (i.e. the number of reproductive records per species). Therefore, the absence of reproductive senescence in e.g. Sea Otter (*Enhydra lutris*) or Orangutan (*Pongo pygmaeus*) might simply be a direct consequence of low sample size ($N = 36$ and $N = 58$ for which the probability to detect senescence was only 0.40 and 0.46, respectively; see *Appendix C* - Figure S2). This shows that we faced a lack of statistical power to detect a statistically significant decline in reproductive performance in late life in some hunted populations (e.g. racoon dog, *Nyctereutes procyonydes*, Helle and Kauhala, 1993). It is also noteworthy that sample size also influenced the shape we identified for the reproductive senescence patterns because the number of segments (from 1 to 3) increased with the sample size (*Appendix C* - Table S5).

Third, we might have failed to detect reproductive senescence in some of the species due to discrepancies between our approach and the approaches used in the original studies. To compare reproductive senescence across mammalian species, we had to apply a standardized approach to estimate parameters of reproductive senescence across all species, which has led to some differences in the data themselves (aggregated data per age in our study vs. individual data in the original study) and also in the way data were analyzed. For instance, in Columbian ground squirrels (*Spermophilus columbianus*), Broussard and colleagues (Broussard et al., 2003) detected reproductive senescence by comparing two groups of females (2-5 years old vs. 6-9 years old), whereas we did not detect reproductive senescence by analyzing the full age-dependent reproductive trajectory. Despite these constraints the species-specific estimates we reported for the onset of reproductive senescence closely matched the information provided in the original source *(Appendix A*, Table S2).

Lastly, there is now widespread evidence that the senescence process is asynchronous among traits (Gaillard and Lemaître, 2017; Promislow et al., 2006). For instance, some immune traits drop sharply with increasing age, while others remain quite constant throughout life (Cheynel et al., 2017). Interestingly, the asynchrony in senescence patterns can also occur among traits within the single reproductive function (Lemaître and Gaillard, 2017). Let compare for instance reproductive senescence in Alpine marmot (*Marmota marmota*) and Soay sheep (*Ovis aries*). In marmots, female litter size decreases with increasing age but offspring mass stays constant with maternal age (Berger et al., 2015b), whereas the opposite is observed in Soay sheep (Hayward et al., 2013). Our analysis mostly focused on the m_x values but we cannot exclude that species in which we did not detect reproductive senescence display age-specific decline in at least another reproductive trait. For instance, a recent meta-analysis demonstrated that a decreasing offspring survival with increasing maternal age is widespread in mammals (Ivimey-Cook and Moorad, 2020).

Overall, current evidence suggests that reproductive senescence is - similarly to actuarial senescence - the rule rather than the exception in mammals. Nevertheless, the shape of reproductive senescence (here depicted by the variable number of segments included in the selected model, *Appendix A*, Table S1) as well as its timing (i.e. age at the onset of reproductive senescence) and intensity (i.e. rate of reproductive senescence) display a high diversity across mammalian species.

The influence of life history, mating behavior and ovulation mode on reproductive senescence

As predicted, we found that both the onset and the rate of reproductive senescence were tightly associated with the age at first reproduction. In absence of data required to estimate generation time, the age at first reproduction constitutes the most reliable proxy of the species position along the slow-fast continuum (Gaillard et al., 2005). As previously acknowledged by Jones et al. (2008), slow species show a later age at reproductive senescence and a lower rate of reproductive senescence compared to fast species. The species ranking according to the pattern of covariation in biological times is the most recognized axis of variation across mammalian species (Gaillard et al., 2016; Stearns, 1983). Since both the onset and the rate of reproductive senescence can be expressed in time units (years and $years^{-1}$, respectively), these two metrics are logically aligned with the other biological times (e.g. generation time, age at first reproduction, lifespan or gestation length). Moreover, as several life history traits measured in time units are known to be strongly influenced by phylogeny (e.g. Capellini et al., 2008 for the sleep duration; Wootton, 1987 for the age at first reproduction, Lemaître et

al. 2020b for adult lifespan), it is not so surprising that both the onset and the rate of reproductive senescence also depend on the species taxonomic position. When the onsets of reproductive senescence are scaled by the maximum lifespan recorded in the focal populations, (i.e. standardized for the species pace of life), the influence of the phylogeny was unsurprisingly strongly reduced.

Once controlled for the species-specific pace of life, we found that traits associated with female mating behavior and physiology influenced the timing of reproductive senescence. Interestingly, the rate of reproductive senescence was not detectably influenced by mating patterns, which adds to the growing evidence that both the onset and the rate of senescence have to be investigated simultaneously when it comes to identify factors influencing senescence patterns. Contrary, to our expectations, the onset of reproductive senescence was earlier in species with induced ovulation than in those with spontaneous ovulation. This might seem surprising at first glance because the intensity of sperm competition (and its possible deleterious consequences for females) are known to be higher in presence of spontaneous ovulation (Iossa et al., 2008; Soulsbury and Iossa, 2010). Yet, two hypotheses can be proposed to explain this unexpected result. First, in many species with induced ovulation, males bear keratinized spines on the surface of the penis (e.g. Dixson, 1991 common marmoset *Callithrix jacchus*; Lemaître et al., 2012 in bank voles, *Myodes glareolus*), which could be involved in the induction of ovulation during the mating (Parag et al., 2006; Stoddart and Milligan, 1979). The density of spines on the male genitalia can in some species be high, with spines being particularly sharp (Orr and Brennan, 2016; Parag et al., 2006; Stockley, 2002). On the long run, repeated injuries caused by spines to the female reproductive tract likely impair female's reproductive condition and might be responsible for the earlier age at the onset of reproductive senescence we reported for these species. While this hypothesis clearly deserves further consideration from studies performed both within and across species, it might not be the whole story, as some species with spontaneous ovulation can also bear spines (e.g. in some primates, see Stockley, 2002). Another (non-mutually exclusive) explanation is that the efficiency of the neuro-endocrinological pathway regulating the relationship between mating and ovulation (reviewed in Bakker and Baum, 2000) decreases with increasing age. So far, the ageing of many physiological functions governing female reproduction has been described in human and laboratory rodents (vom Saal et al., 1994). However, whether neuronal mechanisms controlling the cascade of physiological responses (e.g. release of reproductive hormones by the hypothalamic–pituitary–gonadal axis)

following mating in species with induced ovulation are particularly prone to senesce is currently unknown.

We also found that - independently of the ovulation mode - species where females mate multiply with different males during a given reproductive bout (i.e. when male's relative testes mass is high) also suffer from an earlier onset of reproductive senescence. This contrasts with a previous comparative analysis that revealed that females displaying such behavior do not pay any costs in terms of reduced lifespan or strengthened actuarial senescence (Lemaître and Gaillard, 2013). Overall, this suggests that the physiological costs associated with multiple mating in females are paid in terms of reproductive senescence rather than actuarial senescence. A few hypotheses can be elaborated to explain this link. For instance, evidence that infectious diseases negatively impact reproductive success is increasing (e.g. Nguyen et al., 2015; Pioz et al., 2008). The increased risk of contracting sexually transmitted diseases throughout life in promiscuous females (Nunn et al., 2014) might compromise reproductive performance in late life. In addition, mammalian females might also suffer from the repeated exposure to the male seminal fluid. Such deleterious consequences have been reported in *Drosophila melanogaster*, for which some specific seminal fluid proteins influence various aspects of female reproductive physiology, notably receptivity (see Chapman, 2001 for a review). The composition of the seminal fluid is also complex in mammals (Poiani, 2006) and can impact female reproductive physiology (e.g. by inducing ovulation in some camelids, see Ratto et al., 2010). However, whether male seminal fluid proteins are associated with long-term deleterious effects on female reproductive success is yet to be investigated and constitutes one promising avenue of research.

Future directions

We provide a first evidence of the high diversity of reproductive senescence patterns across mammals in the wild and identify key biological and behavioral factors that shape this diversity. Yet, we are only scratching the surface of the reproductive senescence conundrum and we point out below some of the most promising avenues for future research in this area.

We found a huge diversity of reproductive senescence patterns across mammalian species. Nevertheless, whether this interspecific variation translates to high variation among populations within a given species remains unknown. Indeed, while there is now compelling evidence that environmental conditions modulate age-specific survival patterns, as individuals in captivity have longer lifespan and similar or later onset of actuarial senescence than their wild conspecifics (Tidière et al. 2016), similar large-scale comparative studies are lacking for

553 reproductive senescence. Empirical studies have revealed that the amount of resources
554 available in the environment positively influences the female reproductive output (e.g.
555 Persson, 2005). However, as females age, their ability to maintain large home range (e.g. Froy
556 et al., 2018 in Soay sheep, *Ovis aries* and red deer, *Cervus elaphus*), to acquire resources (e.g.
557 MacNulty et al., 2009 in wolves) or to eat and digest food (e.g. Gaillard et al. 2015 for a study
558 on tooth wear in ruminants) are impaired, which ultimately decreases the efficiency of food
559 provisioning to their progeny during the energy-demanding period of maternal care
560 (Gittleman and Thompson, 1988; Clutton-Brock et al. 1989; see also below) and thereby
561 jeopardizes their reproductive success. However, whether the quality and quantity of
562 resources available in the environment mitigate the reproductive ageing cost of these
563 functional declines remains to be investigated. Moreover, following a resource-based
564 allocation trade-off, a high reproductive effort during early life can be paid in terms of
565 increased reproductive senescence (Douhard et al., 2020; Lemaître et al., 2015; Nussey et al.,
566 2006). For instance, it has been suggested that the early onset of reproductive senescence
567 reported in domestic mammals might be due to the strong artificial selection for a high
568 reproductive effort during early life in domestic animals (Mysterud et al., 2002 for sheep,
569 Grange et al. 2009 for horses, *Equus caballus*). Alternatively, whether these costs are rather
570 induced by a mismatch with environmental conditions remains unknown. In that context,
571 comparing mammalian populations facing contrasted environmental conditions would allow
572 assessing whether some species simply escape reproductive senescence or whether
573 particularly good environmental conditions prevent reproductive senescence to occur.

574 While we found that female mating behavior, ovulation mode, allometry and pace of
575 life all shape reproductive senescence, many other biological factors are likely to influence
576 either its onset or its rate. It is beyond the scope to list all of them but we can emphasize two
577 aspects we consider as being particularly promising: the cost of lactation and the type of
578 placentation. In mammals, the lactation period constitutes the most energy-demanding part of
579 the reproductive cycle (Clutton-Brock et al., 1989) and to produce high quality milk in
580 sufficient quantity, females need to increase their food intake. Interestingly, the magnitude of
581 this extra food intake varies substantially across species (see Douhard et al., 2016; Hayssen
582 and Orr, 2017 for reviews), and the energy intake required by lactating females can
583 sometimes be two- or threefold that required during the non-breeding period (see Speakman,
584 2008 for some striking examples in rodents and insectivores). The physiological costs
585 associated to lactation are diverse and well-documented, ranging from organ remodeling (e.g.
586 increased size of liver, pancreas and mammary glands) to a risk of hyperthermia or bone loss

(due to the need to transfer calcium to the offspring) (Dufour and Sauther, 2002; Jasienska, 2013; Speakman, 2008). On the contrary, much less is known on the long-term fitness costs of lactation, notably in terms of ageing. We hypothesize that the repeated physiologically costly periods of lactation should translate into an earlier/and or stronger reproductive senescence. Moreover, such costs are likely to be buffered or exacerbated by the quality of the environment, which in turn might constitute selective pressures towards specific reproductive tactics. For instance, the harsh environmental conditions faced by female white-tailed deer (*Odocoileus virginianus*) in Minnesota (i.e. severe winters and wolf predation) might explain their relatively low allocation to reproduction throughout their lives, a tactic that might ultimately minimize reproductive senescence as reported by DelGuidice and colleagues (DelGiudice et al., 2007) in this population. Another interesting feature of the mammalian reproductive machinery is the structure of the placenta that can influence the amount of nutrients transferred by the mother to the fetus(es). Indeed, the morphology of the placenta is highly variable across species, notably in its degree of invasiveness and interdigitation (Wildman, 2011), which ultimately modulates the degree and the surface for nutrient transfer during gestation (Capellini et al., 2011). Therefore, the placenta constitutes a critical organ for parent-offspring conflicts (Haig, 1993). It has already been observed that the embryo growth rate is approximately twice as fast in more interdigitated placentas (Capellini et al., 2011) and that placenta types covary with the pace of life. Slow-living species are generally characterized by an invasive placenta (i.e. hemochorial placenta) while fast species are most often characterized by less invasive placenta types (i.e. endotheliochorial or epitheliochorial placenta) (Garratt et al., 2013). The evolution of a less invasive placenta has allowed mothers to limit fetal manipulation (i.e. when food transfer during gestation is largely under the fetus(es) control) and to control better food provisioning among offspring and across reproductive events (Garratt et al., 2013). Therefore - everything else being equal - such placenta types might confer to females a way to buffer reproductive senescence.

As mentioned earlier, our work focused on female reproductive rate, as females concentrate the huge majority of the data published so far. However, recent studies have highlighted the evolutionary relevance of studying male reproductive senescence (Lemaître and Gaillard, 2017; Monaghan and Metcalfe, 2019), as well as its potential medical implications (Levine et al., 2018). As a consequence, evidence that male reproductive senescence is pervasive in the wild (reviewed in Lemaître and Gaillard, 2017) is accumulating. Similar to what we have discussed above for females, male reproductive senescence can be assessed through a large variety of traits, notably an age-specific decline in

secondary sexual traits, ejaculate quality, mating rate or reproductive success. However, to be directly comparable, male and female reproductive senescence need to be investigated on the same trait and the potential confounding effects of the partner's age need to be accounted for (Thorley et al., 2020). Surprisingly, we still lack a comprehensive theoretical framework for the evolution of sex differences in reproductive senescence. However, following our current life history framework, we can expect polygynous mammals where males disproportionately allocate to sexual competition to display most strongly male-biased reproductive senescence. Data available so far suggest that this scenario might be true. In the monogamous meerkat (*Suricata suricatta*), no sex difference in reproductive senescence has been detected (Thorley et al., 2020) while in polygynous red deer (*Cervus elaphus*), the rate of reproductive senescence (measured through annual fecundity) is much steeper in males than in females (Nussey et al., 2009; see also Muller et al., 2020 in chimpanzee, *Pan troglodytes schweinfurthii* but see Festa-Bianchet, 2012 for some counter-examples, e.g. Bighorn sheep, *Ovis canadensis*, in Ram mountain). Studies investigating these questions in a wider range of species are now required.

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Author contributions

JFL and JMG designed the study. JFL, JMG and VR collected, analyzed and wrote the manuscript.

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Declaration of Competing Interest

The authors declare no conflict of interest

660 **References**

661 Alberts, S.C., Altmann, J., Brockman, D.K., Cords, M., Fedigan, L.M., Pusey, A., Stoinski,
662 T.S., Strier, K.B., Morris, W.F., Bronikowski, A.M., 2013. Reproductive aging patterns
663 in primates reveal that humans are distinct. Proceedings of the National Academy of
664 Sciences 110, 13440–13445.

665 Arnqvist, G., Rowe, L., 2005. Sexual conflict. Princeton University Press.

666 Bakker, J., Baum, M.J., 2000. Neuroendocrine regulation of GnRH release in induced
667 ovulators. Frontiers in neuroendocrinology 21, 220–262.

668 Baudisch, A., Stott, I., 2019. A pace and shape perspective on fertility. Methods in Ecology
669 and Evolution 10, 1941–1951.

670 Berger, V., Lemaître, J.-F., Gaillard, J.-M., Cohas, A., 2015a. How do animals optimize the
671 size–number trade-off when aging? Insights from reproductive senescence patterns in
672 marmots. Ecology 96, 46–53.

673 Berger, V., Lemaître, J.-F., Gaillard, J.-M., Cohas, A., 2015b. How do animals optimize the
674 size–number trade-off when aging? Insights from reproductive senescence patterns in
675 marmots. Ecology 96, 46–53.

676 Berman, M., Gaillard, J.-M., Weimerskirch, H., 2009. Contrasted patterns of age-specific
677 reproduction in long-lived seabirds. Proceedings of the Royal Society B: Biological
678 Sciences 276, 375–382.

679 Bininda-Emonds, O.R., Cardillo, M., Jones, K.E., MacPhee, R.D., Beck, R.M., Grenyer, R.,
680 Price, S.A., Vos, R.A., Gittleman, J.L., Purvis, A., 2008. Erratum: The delayed rise of
681 present-day mammals. Nature 456, 274–274.

682 Bininda-Emonds, O.R., Cardillo, M., Jones, K.E., MacPhee, R.D., Beck, R.M., Grenyer, R.,
683 Price, S.A., Vos, R.A., Gittleman, J.L., Purvis, A., 2007. The delayed rise of present-
684 day mammals. Nature 446, 507.

685 Bronikowski, A.M., Altmann, J., Brockman, D.K., Cords, M., Fedigan, L.M., Pusey, A.,
686 Stoinski, T., Morris, W.F., Strier, K.B., Alberts, S.C., 2011. Aging in the natural world:
687 comparative data reveal similar mortality patterns across primates. Science 331, 1325–
688 1328.

689 Broussard, D.R., Risch, T.S., Dobson, F.S., Murie, J.O., 2003. Senescence and age-related
690 reproduction of female Columbian ground squirrels. Journal of Animal Ecology 72,
691 212–219.

692 Brunet-Rossinni, A.K., Austad, S.N., 2006. Senescence in wild populations of mammals and
693 birds, in: Handbook of the Biology of Aging (Sixth Edition). Elsevier, pp. 243–266.

694 Burnham, K.P., Anderson, D.R., 2003. Model selection and multimodel inference: a practical
695 information-theoretic approach. Springer Science & Business Media.

696 Capellini, I., Barton, R.A., McNamara, P., Preston, B.T., Nunn, C.L., 2008. Phylogenetic
697 analysis of the ecology and evolution of mammalian sleep. Evolution: International
698 Journal of Organic Evolution 62, 1764–1776.

699 Capellini, I., Venditti, C., Barton, R.A., 2011. Placentation and maternal investment in
700 mammals. The American Naturalist 177, 86–98.

701 Caughley, G., 1976. Wildlife management and the dynamics of ungulate populations. In
702 'Applied Biology'.(Ed. TH Coaker.) pp. 183–246. Academic Press: New York.

703 Caughley, G., 1966. Mortality patterns in mammals. Ecology 47, 906–918.

704 Chapman, T., 2001. Seminal fluid-mediated fitness traits in Drosophila. Heredity 87, 511–
705 521.

706 Cheynel, L., Lemaître, J.-F., Gaillard, J.-M., Rey, B., Bourgoin, G., Ferté, H., Jégo, M.,
707 Débias, F., Pellerin, M., Jacob, L., 2017. Immunosenescence patterns differ between
708 populations but not between sexes in a long-lived mammal. Scientific reports 7, 13700.

709 Clutton-Brock, T.H., Albon, S.D., Guinness, F.E., 1989. Fitness costs of gestation and
710 lactation in wild mammals. Nature 337, 260.

711 Cohen, A.A., 2004. Female post-reproductive lifespan: a general mammalian trait. Biological
712 Reviews 79, 733–750.

713 Cohen, A.A., Kennedy, B.K., Anglas, U., Bronikowski, A.M., Deelen, J., Dufour, F.,
714 Ferbeyre, G., Ferrucci, L., Franceschi, C., Frasca, D., *et al.* 2020. Lack of consensus on
715 an aging biology paradigm? A global survey reveals an agreement to disagree, and the
716 need for an interdisciplinary framework. Mechanisms of ageing and development 191,
717 111316.

718 Colchero, F., Jones, O.R., Conde, D.A., Hodgson, D., Zajitschek, F., Schmidt, B.R., Malo,
719 A.F., Alberts, S.C., Becker, P.H., Bouwhuis, S., 2019. The diversity of population
720 responses to environmental change. Ecology letters 22, 342–353.

721 Coulson, J.C., Horobin, J., 1976. The influence of age on the breeding biology and survival of
722 the Arctic Tern Sterna paradisaea. Journal of Zoology 178, 247–260.

723 DelGiudice, G.D., Lenarz, M.S., Powell, M.C., 2007. Age-specific fertility and fecundity in
724 northern free-ranging white-tailed deer: evidence for reproductive senescence? Journal
725 of Mammalogy 88, 427–435.

726 Dixson, A.F., 1991. Penile spines affect copulatory behaviour in a primate (*Callithrix*
727 *jacchus*). Physiology & behavior 49, 557–562.

728 Dormann, C.F., Elith, J., Bacher, S., Buchmann, C., Carl, G., Carré, G., Marquéz, J.R.G.,
729 Gruber, B., Lafourcade, B., Leitão, P.J., 2013. Collinearity: a review of methods to deal
730 with it and a simulation study evaluating their performance. Ecography 36, 27–46.

731 Douhard, F., Lemaître, J.-F., Rauw, W.M., Friggens, N.C., 2016. Allometric scaling of the
732 elevation of maternal energy intake during lactation. Frontiers in Zoology 13.

733 Douhard, M., Festa-Bianchet, M., Pelletier, F., 2020. Sons accelerate maternal aging in a wild
734 mammal. Proceedings of the National Academy of Sciences 117, 4850–4857.

735 Dufour, D.L., Sauther, M.L., 2002. Comparative and evolutionary dimensions of the
736 energetics of human pregnancy and lactation. American Journal of Human Biology 14,
737 584–602.

738 Ellis, S., Franks, D.W., Nattrass, S., Cant, M.A., Bradley, D.L., Giles, D., Balcomb, K.C.,
739 Croft, D.P., 2018. Postreproductive lifespans are rare in mammals. Ecology and
740 evolution 8, 2482–2494.

741 Emlen, J.M., 1970. Age specificity and ecological theory. Ecology 51, 588–601.

742 Felsenstein, J., 1985. Phylogenies and the comparative method. The American Naturalist 125,
743 1–15.

744 Festa-Bianchet, M., 2012. The cost of trying: weak interspecific correlations among life-
745 history components in male ungulates. Canadian Journal of Zoology 90, 1072–1085.

Freckleton, R.P., Harvey, P.H., Pagel, M., 2002. Phylogenetic analysis and comparative data: a test and review of evidence. The American Naturalist 160, 712–726.

Fritz, S.A., Purvis, A., 2010. Selectivity in mammalian extinction risk and threat types: a new measure of phylogenetic signal strength in binary traits. Conservation Biology 24, 1042–1051.

Froy, H., Börger, L., Regan, C.E., Morris, A., Morris, S., Pilkington, J.G., Crawley, M.J., Clutton-Brock, T.H., Pemberton, J.M., Nussey, D.H., 2018. Declining home range area predicts reduced late-life survival in two wild ungulate populations. Ecology letters 21, 1001–1009.

Froy, H., Lewis, S., Nussey, D.H., Wood, A.G., Phillips, R.A., 2017. Contrasting drivers of reproductive ageing in albatrosses. Journal of Animal Ecology 86, 1022–1032.

Gage, M.J., Freckleton, R.P., 2003. Relative testis size and sperm morphometry across mammals: no evidence for an association between sperm competition and sperm length. Proceedings of the Royal Society of London. Series B: Biological Sciences 270, 625–632.

Gaillard, J.-M., Allaine, D., Pontier, D., Yoccoz, N.G., Promislow, D.E., 1994. Senescence in natural populations of mammals: a reanalysis. Evolution 48, 509–516.

Gaillard, J.-M., Lemaître, J.-F., 2020. An integrative view of senescence in nature. Functional Ecology 34, 4–16.

Gaillard, J.-M., Lemaître, J.-F., 2017. The Williams' legacy: A critical reappraisal of his nine predictions about the evolution of senescence. Evolution 71, 2768–2785.

Gaillard, J.-M., Lemaitre, J.F., Berger, V., Bonenfant, C., Devillard, S., 2016. Axes of variation in life histories. The Encyclopedia of Evolutionary Biology: Elsevier 312–23.

Gaillard, J.-M., Yoccoz, N.G., Lebreton, J.-D., Bonenfant, C., Devillard, S., Loison, A., Pontier, D., Allaine, D., 2005. Generation time: a reliable metric to measure life-history variation among mammalian populations. The American Naturalist 166, 119–123.

Garratt, M., Gaillard, J.-M., Brooks, R.C., Lemaitre, J.-F., 2013. Diversification of the eutherian placenta is associated with changes in the pace of life. Proceedings of the National Academy of Sciences 110, 7760–7765.

Gittleman, J.L., Thompson, S.D., 1988. Energy allocation in mammalian reproduction. American zoologist 28, 863–875.

Gomendio, M., Harcourt, A.H, Roldan, Eduardo, 1998. Sperm competition in mammals. In S*perm competition and sexual selection* (Edited by T.R. Birkhead & A.P. Moller). Academic Press.

Grange, S., Duncan, P., Gaillard, J.-M., 2009. Poor horse traders: large mammals trade survival for reproduction during the process of feralization. Proceedings of the Royal Society B: Biological Sciences 276, 1911–1919.

Haig, D., 1993. Genetic conflicts in human pregnancy. The Quarterly review of biology 68, 495–532.

Hamlin, K.L., Pac, D.F., Sime, C.A., DeSimone, R.M., Dusek, G.L., 2000. Evaluating the accuracy of ages obtained by two methods for Montana ungulates. The Journal of Wildlife Management 441–449.

Harcourt, H., Purvis, A., Liles, L., 1995. Sperm competition: mating system, not breeding

789 season, affects testes size of primates. Functional Ecology 468–476.

790 Hayssen, V., Orr, T.J., 2017. Reproduction in mammals: the female perspective. JHU Press.

791 Hayward, A.D., Wilson, A.J., Pilkington, J.G., Clutton-Brock, T.H., Pemberton, J.M., Kruuk,
792 L.E., 2013. Reproductive senescence in female Soay sheep: variation across traits and
793 contributions of individual ageing and selective disappearance. Functional Ecology 27,
794 184–195.

795 Helle, E., Kauhala, K., 1993. Age structure, mortality, and sex ratio of the raccoon dog in
796 Finland. Journal of Mammalogy 74, 936–942.

797 Iossa, G., Soulsbury, C.D., Baker, P.J., Harris, S., 2008. Sperm competition and the evolution
798 of testes size in terrestrial mammalian carnivores. Functional Ecology 22, 655–662.

799 Ives, A.R., Garland Jr, T., 2010. Phylogenetic logistic regression for binary dependent
800 variables. Systematic biology 59, 9–26.

801 Ivimey-Cook, E., Moorad, J., 2020. The diversity of maternal-age effects upon pre-adult
802 survival across animal species. Proceedings of the Royal Society B 287, 20200972.

803 Jasienska, G., 2013. The fragile wisdom: an evolutionary view on women's biology and
804 health. Harvard University Press.

805 Jennions, M.D., Petrie, M., 2000. Why do females mate multiply? A review of the genetic
806 benefits. Biological Reviews 75, 21–64.

807 Jones, O.R., Gaillard, J.-M., Tuljapurkar, S., Alho, J.S., Armitage, K.B., Becker, P.H., Bize,
808 P., Brommer, J., Charmantier, A., Charpentier, M., 2008. Senescence rates are
809 determined by ranking on the fast–slow life-history continuum. Ecology Letters 11,
810 664–673.

811 Jones, O.R., Scheuerlein, A., Salguero-Gómez, R., Camarda, C.G., Schaible, R., Casper, B.B.,
812 Dahlgren, J.P., Ehrlén, J., García, M.B., Menges, E.S., 2014. Diversity of ageing across
813 the tree of life. Nature 505, 169.

814 Kamilar, J.M., Cooper, N., 2013. Phylogenetic signal in primate behaviour, ecology and life
815 history. Phil. Trans. R. Soc. B 368, 20120341.

816 Karniski, C., Krzyszczyk, E., Mann, J., 2018. Senescence impacts reproduction and maternal
817 investment in bottlenose dolphins. Proc. R. Soc. B 285, 20181123.

818 Kasuya, T., Marsh, H., 1984. Life history and reproductive biology of the short-finned pilot
819 whale, Globicephala macrorhynchus, off the Pacific coast of Japan. Report of the
820 International Whaling Commission, Special 6, 259–310.

821 Le Boeuf, B.J., Laws, R.M., 1994. Elephant seals: population ecology, behavior, and
822 physiology. Univ of California Press.

823 Lee, P.C., Fishlock, V., Webber, C.E., Moss, C.J., 2016. The reproductive advantages of a
824 long life: longevity and senescence in wild female African elephants. Behavioral
825 ecology and sociobiology 70, 337–345.

826 Lemaître, J.-F., Berger, V., Bonenfant, C., Douhard, M., Gamelon, M., Plard, F., Gaillard, J.-
827 M., 2015. Early-late life trade-offs and the evolution of ageing in the wild, in: Proc. R.
828 Soc. B. p. 20150209.

829 Lemaître, J.-F., Gaillard, J.-M., 2017. Reproductive senescence: new perspectives in the wild:
830 Reproductive senescence in the wild. Biological Reviews 92, 2182–2199.

Lemaître, J.-F., Gaillard, J.-M., 2013. Polyandry Has No Detectable Mortality Cost in Female Mammals. PLoS ONE 8, e66670. https://doi.org/10.1371/journal.pone.0066670

Lemaître, J.-F., Gaillard, J.-M., Lackey, L.B., Clauss, M., Müller, D.W.H., 2013. Comparing free-ranging and captive populations reveals intra-specific variation in aging rates in large herbivores. Experimental Gerontology 48, 162–167.

Lemaître, J.-F., Gaillard, J.-M., Ramm, S.A., 2020a. The hidden ageing costs of sperm competition. Ecology Letters. https://doi.org/10.1111/ele.13593

Lemaître, J.-F., Garratt, M., Gaillard, J.-M., 2020b. Going beyond Lifespan in Comparative Biology of Aging. Advances in Geriatric Medicine and Research 2.

Lemaître, J.-F., Ramm, S.A., Barton, R.A., Stockley, P., 2009. Sperm competition and brain size evolution in mammals. Journal of Evolutionary Biology 22, 2215–2221. https://doi.org/10.1111/j.1420-9101.2009.01837.x

Lemaître, J.-F., Ramm, S.A., Jennings, N., Stockley, P., 2012. Genital morphology linked to social status in the bank vole (*Myodes glareolus*). Behavioral Ecology and Sociobiology 66, 97–105. https://doi.org/10.1007/s00265-011-1257-4

Lemaître, J.-F., Ronget, V., Tidière, M., Allainé, D., Berger, V., Cohas, A., Colchero, F., Conde, D.A., Garratt, M., Liker, A., 2020c. Sex differences in adult lifespan and aging rates of mortality across wild mammals. Proceedings of the National Academy of Sciences 117, 8546–8553.

Leslie, P.H., Ranson, R.M., 1940. The mortality, fertility and rate of natural increase of the vole (*Microtus agrestis*) as observed in the laboratory. The Journal of Animal Ecology 27–52.

Levine, H., Mohri, H., Ekbom, A., Ramos, L., Parker, G., Roldan, E., Jovine, L., Koelle, S., Lindstrand, A., Immler, S., Mortimer, S., Mortimer, D., van der Horst, G., Ishijima, S., Aneck-Hahn, N., Baldi, E., Menkveld, R., Rothmann, S.A., Giwercman, A., Giwercman, Y., Holmberg, M., Kvist, U., Björndahl, L., Holmberg, R., Arver, S., Flanagan, J., Drevet, J.R., 2018. Male reproductive health statement (XIIIth international symposium on Spermatology, may 9th–12th 2018, Stockholm, Sweden. Basic and Clinical Andrology 28, 13. https://doi.org/10.1186/s12610-018-0077-z

Lieury, N., Drouet-Hoguet, N., Ruette, S., Devillard, S., Albaret, M., Millon, A., 2017. Rural populations of the red fox Vulpes vulpes show little evidence of reproductive senescence. Mammalian biology 87, 146–151.

Link, W.A., Barker, R.J., 2006. Model weights and the foundations of multimodel inference. Ecology 87, 2626–2635.

Lüpold, S., 2013. Ejaculate quality and constraints in relation to sperm competition levels among eutherian mammals. Evolution 67, 3052–3060.

MacNulty, D.R., Smith, D.W., Vucetich, J.A., Mech, L.D., Stahler, D.R., Packer, C., 2009. Predatory senescence in ageing wolves. Ecology letters 12, 1347–1356.

Monaghan, P., Charmantier, A., Nussey, D.H., Ricklefs, R.E., 2008. The evolutionary ecology of senescence. Functional Ecology 22, 371–378.

Monaghan, P., Metcalfe, N.B., 2019. The deteriorating soma and the indispensable germline: gamete senescence and offspring fitness. Proc. R. Soc. Lond. B 286, 20192187.

Muggeo, V.M., 2017. Interval estimation for the breakpoint in segmented regression: a smoothed score-based approach. Australian & New Zealand Journal of Statistics 59,

311–322.

Nguyen, N., Fashing, P.J., Boyd, D.A., Barry, T.S., Burke, R.J., Goodale, C.B., Jones, S.C., Kerby, J.T., Kellogg, B.S., Lee, L.M., 2015. Fitness impacts of tapeworm parasitism on wild gelada monkeys at Guassa, Ethiopia. American journal of primatology 77, 579–594.

Nunn, C.L., Scully, E.J., Kutsukake, N., Ostner, J., Schülke, O., Thrall, P.H., 2014. Mating competition, promiscuity, and life history traits as predictors of sexually transmitted disease risk in primates. International Journal of Primatology 35, 764–786.

Nussey, D.H., Froy, H., Lemaitre, J.-F., Gaillard, J.-M., Austad, S.N., 2013. Senescence in natural populations of animals: widespread evidence and its implications for bio-gerontology. Ageing research reviews 12, 214–225.

Nussey, D.H., Kruuk, L.E., Donald, A., Fowlie, M., Clutton-Brock, T.H., 2006. The rate of senescence in maternal performance increases with early-life fecundity in red deer. Ecology Letters 9, 1342–1350.

Nussey, D.H., Kruuk, L.E., Morris, A., Clements, M.N., Pemberton, J.M., Clutton-Brock, T.H., 2009. Inter-and intrasexual variation in aging patterns across reproductive traits in a wild red deer population. The American Naturalist 174, 342–357.

Ollason, J.C., Dunnet, G.M., 1978. Age, experience and other factors affecting the breeding success of the Fulmar, *Fulmarus glacialis*, in Orkney. The Journal of Animal Ecology 961–976.

Orme, D., Freckleton, R., Thomas, G., Petzoldt, T., Fritz, S., 2013. The caper package: comparative analysis of phylogenetics and evolution in R. R package version 5, 1–36.

Orr, T.J., Brennan, P.L., 2016. All features great and small—the potential roles of the baculum and penile spines in mammals. Integrative and comparative biology 56, 635–643.

Pagel, M., 1999. Inferring the historical patterns of biological evolution. Nature 401, 877–884.

Paradis, E., Claude, J., Strimmer, K., 2004. APE: analyses of phylogenetics and evolution in R language. Bioinformatics 20, 289–290.

Parag, A., Bennett, N.C., Faulkes, C.G., Bateman, P.W., 2006. Penile morphology of African mole rats (Bathyergidae): structural modification in relation to mode of ovulation and degree of sociality. Journal of Zoology 270, 323–329.

Parker, G.A., 2016. The evolution of expenditure on testes. Journal of Zoology 298, 3–19.

Parker, G.A., 1970. Sperm competition and its evolutionary consequences in the insects. Biological Reviews 45, 525–567.

Péron, G., Bonenfant, C., Lemaitre, J.-F., Ronget, V., Tidiere, M., Gaillard, J.-M., 2019a. Does grandparental care select for a longer lifespan in non-human mammals? Biological Journal of the Linnean Society 128, 360–372.

Péron, G., Lemaître, J.-F., Ronget, V., Tidière, M., Gaillard, J.-M., 2019b. Variation in actuarial senescence does not reflect life span variation across mammals. PLoS biology 17, e3000432.

Perrins, C.M., Moss, D., 1974. Survival of young Great Tits in relation to age of female parent. Ibis 116, 220–224.

Persson, J., 2005. Female wolverine (*Gulo gulo*) reproduction: reproductive costs and winter food availability. Canadian Journal of Zoology 83, 1453–1459.

Peters, A., Delhey, K., Nakagawa, S., Aulsebrook, A., Verhulst, S., 2019. Immunosenescence in wild animals: meta-analysis and outlook. Ecology letters.

Photopoulou, T., Ferreira, I.M., Best, P.B., Kasuya, T., Marsh, H., 2017. Evidence for a postreproductive phase in female false killer whales Pseudorca crassidens. Frontiers in zoology 14, 30.

Pioz, M., Loison, A., Gauthier, D., Gibert, P., Jullien, J.-M., Artois, M., Gilot-Fromont, E., 2008. Diseases and reproductive success in a wild mammal: example in the alpine chamois. Oecologia 155, 691–704.

Poiani, A., 2006. Complexity of seminal fluid: a review. Behavioral Ecology and Sociobiology 60, 289–310.

Promislow, D.E., 1991. Senescence in natural populations of mammals: a comparative study. Evolution 45, 1869–1887.

Promislow, D.E., Fedorka, K.M., Burger, J.M., 2006. Evolutionary biology of aging: future directions, in: Handbook of the Biology of Aging. Elsevier, pp. 217–242.

Ramm, S.A., Parker, G.A., Stockley, P., 2005. Sperm competition and the evolution of male reproductive anatomy in rodents. Proceedings of the Royal Society of London B: Biological Sciences 272, 949–955.

Ratto, M.H., Huanca, W., Adams, G.P., 2010. Ovulation-inducing factor: a protein component of llama seminal plasma. Reproductive Biology and Endocrinology 8, 44.

Ricklefs, R.E., 2010. Life-history connections to rates of aging in terrestrial vertebrates. Proceedings of the National Academy of Sciences 107, 10314–10319.

Ricklefs, R.E., 2006. Embryo development and ageing in birds and mammals. Proceedings of the Royal Society B: Biological Sciences 273, 2077–2082.

Ricklefs, R.E., 1998. Evolutionary theories of aging: confirmation of a fundamental prediction, with implications for the genetic basis and evolution of life span. The American Naturalist 152, 24–44.

Ricklefs, R.E., Scheuerlein, A., 2001. Comparison of aging-related mortality among birds and mammals. Experimental gerontology 36, 845–857.

Ronget, V., Gaillard, J.-M., 2020. Assessing ageing patterns for comparative analyses of mortality curves: Going beyond the use of maximum longevity. Functional Ecology.

Sinclair, A.R.E., 1977. The African buffalo; a study of resource limitation of populations.

Soulsbury, C.D., 2010. Genetic patterns of paternity and testes size in mammals. PLoS One 5, e9581.

Soulsbury, C.D., Iossa, G., 2010. The impact of ovulation mode on sperm quantity and quality in mammals. Evolutionary ecology 24, 879–889.

Sparkman, A.M., Blois, M., Adams, J., Waits, L., Miller, D.A., Murray, D.L., 2017. Evidence for sex-specific reproductive senescence in monogamous cooperatively breeding red wolves. Behavioral ecology and sociobiology 71, 6.

Speakman, J.R., 2008. The physiological costs of reproduction in small mammals. Philosophical Transactions of the Royal Society B: Biological Sciences 363, 375–398.

https://doi.org/10.1098/rstb.2007.2145

Stahler, D.R., MacNulty, D.R., Wayne, R.K., VonHoldt, B., Smith, D.W., 2013. The adaptive value of morphological, behavioural and life-history traits in reproductive female wolves. Journal of Animal Ecology 82, 222–234.

Stearns, S.C., 1983. The influence of size and phylogeny on patterns of covariation among life-history traits in the mammals. Oikos 173–187.

Stockley, P., 2003. Female multiple mating behaviour, early reproductive failure and litter size variation in mammals. Proceedings of the Royal Society of London B: Biological Sciences 270, 271–278.

Stockley, P., 2002. Sperm competition risk and male genital anatomy: comparative evidence for reduced duration of female sexual receptivity in primates with penile spines. Evolutionary Ecology 16, 123–137.

Stockley, P., 1997. Sexual conflict resulting from adaptations to sperm competition. Trends in Ecology & Evolution 12, 154–159.

Stoddart, D.M., Milligan, S.R., 1979. The copulatory pattern of the bank vole (Clethrionomys glareolus) and speculation on the role of penile spines. Journal of Zoology 188, 279–283.

Symonds, M.R., Blomberg, S.P., 2014. A primer on phylogenetic generalised least squares, in: Modern Phylogenetic Comparative Methods and Their Application in Evolutionary Biology. Springer, pp. 105–130.

Thorley, J., Duncan, C., Sharp, S.P., Gaynor, D., Manser, M.B., Clutton-Brock, T., 2020. Sex-independent senescence in a cooperatively breeding mammal. Journal of Animal Ecology 89, 1080–1093.

Tidière, M., Gaillard, J.-M., Berger, V., Müller, D.W.H., Bingaman Lackey, L., Gimenez, O., Clauss, M., Lemaître, J.-F., 2016. Comparative analyses of longevity and senescence reveal variable survival benefits of living in zoos across mammals. Scientific Reports 6. https://doi.org/10.1038/srep36361

Tidière, M., Gaillard, J.-M., Müller, D.W., Lackey, L.B., Gimenez, O., Clauss, M., Lemaître, J.-F., 2015. Does sexual selection shape sex differences in longevity and senescence patterns across vertebrates? A review and new insights from captive ruminants. Evolution 69, 3123–3140.

Tietze, C., 1957. Reproductive span and rate of reproduction among Hutterite women. Obstetrical & Gynecological Survey 12, 727–728.

vom Saal, F.S., Finch, C.E., Nelson, J.F., 1994. Natural history and mechanisms of reproductive aging in humans, laboratory rodents, and other selected vertebrates. The physiology of reproduction 2, 1213–1314.

Wildman, D.E., 2011. Toward an integrated evolutionary understanding of the mammalian placenta. Placenta 32, S142–S145.

Wootton, J.T., 1987. The effects of body mass, phylogeny, habitat, and trophic level on mammalian age at first reproduction. Evolution 41, 732–749.

Table 1: Phylogenetically controlled comparative analyses of the probability to detect reproductive senescence and on factors influencing both the onset and the rate of reproductive senescence. This table provides parameter estimates from the models discussed in the text (statistically significant effects occur in bold). For each model displayed below, the estimates are not scaled.

	Dependent variables	Independent variables	Slope ± SE	t	λ [95% CI]	*N*	R^2
a	Probability to detect reproductive senescence	Intercept	1.29 ± 0.40	3.22	NA	101	NA
		Sample size	**0.36 ± 0.15**	**2.32**			
		Hunting status	**-1.24 ± 0.55**	**-1.72**			
		Data quality	-0.71 ± 0.41	-1.72			
b	Onset of reproductive senescence	Intercept	0.78 ± 0.59	1.31	0.75 [NA; 0.93]	69	0.4
		Age at first reproduction	**0.45 ± 0.12**	**3.68**			
		Female body mass	**0.10 ± 0.04**	**2.19**			
c	Onset of reproductive senescence	Intercept	0.95 ± 0.51	1.85	0.63 [NA; 0.91]	69	0.5
		Age at first reproduction	**0.52 ± 0.12**	**4.39**			
		Female body mass	0.08 ± 0.04	1.92			
		Hunting status	0.26 ± 0.16	1.6			
		Data quality	**-0.28 ± 0.13**	**-2.19**			
d	Onset of reproductive senescence	Intercept	0.42 ± 0.47	0.91	0 [NA; 0.80]	54	0.704
		Age at first reproduction	**0.62 ± 0.13**	**5.05**			
		Testes mass	-0.12 ± 0.07	-1.75			
		Male body mass	**0.16 ± 0.07**	**2.35**			
e	Onset of reproductive senescence	Intercept	1.70 ± 0.13	13.04	0 [NA; 0.367]	40	0.74
		Age at first reproduction	**0.71 ± 0.09**	**7.96**			
		Ovulation mode	**-0.50 ± 0.18**	**-2.71**			
f	Onset of reproductive senescence	Intercept	0.45 ± 0.55	0.81	0 [NA; 0.45]	34	0.78
		Age at first reproduction	**0.53 ± 0.13**	**4.11**			
		Ovulation mode	**-0.74 ± 0.22**	**-3.38**			
		Testes mass	**-0.25 ± 0.10**	**-2.44**			
		Male body mass	**0.23 ± 0.09**	**2.61**			
g	Rate of reproductive senescence	Intercept	0.81 ± 0.96	0.85	0.58 [0.18; 0.86]	69	0.49
		Age at first reproduction	**-0.64 ± 0.23**	**-2.78**			
		Female body mass	**-0.31 ± 0.08**	**-3.85**			

Captions for figures

Figure 1: Phylogenetic tree of the species analyzed. The inner band displays the occurrence of reproductive senescence (i.e. presence vs. absence), the middle band displays the *absolute* onset of reproductive senescence (in years) and the outer band displays the *relative* onset of reproductive senescence (absolute onset of reproductive senescence divided by the age of the oldest individual sampled in the population, in proportion).

Figure 2: Age-specific m_x in Bighorn sheep, *Ovis canadensis* at Ram mountain. The fit of the selected two-threshold segmented model is presented. There are three distinct phases in the age-specific reproductive trajectory. The section (A) corresponds to the increasing phase of the reproductive output for young adult females. The section (B) corresponds to a phase for prime-age females during which m_x slightly increases and the section (C) corresponds to a senescent phase characterized by both an onset and a rate of reproductive senescence.

Figure 3: Relationship between the rate of reproductive senescence and the onset of reproductive senescence (on a log-log scale) (Slope of -0.81 ± 0.22; $R^2 = 0.17$; N=69 species).

Figure 4: Relationship between the age at the onset of reproductive senescence (a) or the rate of reproductive senescence (c), and the age at first reproduction (on a log-log scale) (N=69 species). In panels (a) and (c) data are represented independently of female body mass while in panels (b) and (d) data are split according to the female body mass category (light, N= 34 species, heavy, N = 35 species in each body mass category).

Figure 5: Relationship between the onset of reproductive senescence and the relative testes mass (N=54 species). The onset of reproductive senescence corresponds to the residuals of the regression of the onset of reproductive senescence against the age at first reproduction (on a log-log scale). The relative testes mass corresponds to the residuals of the regression of the testes mass against the male body mass (on a log-log scale). In this model, the influence of the phylogeny is not statistically different from 0 ($\lambda = 0$ [NA, 0.80]) and the residuals of the models were thus computed with Ordinary Least Square regressions.

Figure 6: Box-plot displaying the differences in the onset of reproductive senescence between species with spontaneous (N= 28) and induced (N = 12) ovulation. The onset of reproductive senescence corresponds to the residuals of the regression of the onset of reproductive senescence against the age at first reproduction (on a log-log scale, N=40 species). In this model, the influence of the phylogeny is not statistically different from 0 ($\lambda = 0$ [NA, 0.37]) and the residuals of the models were thus computed with Ordinary Least Square regressions.

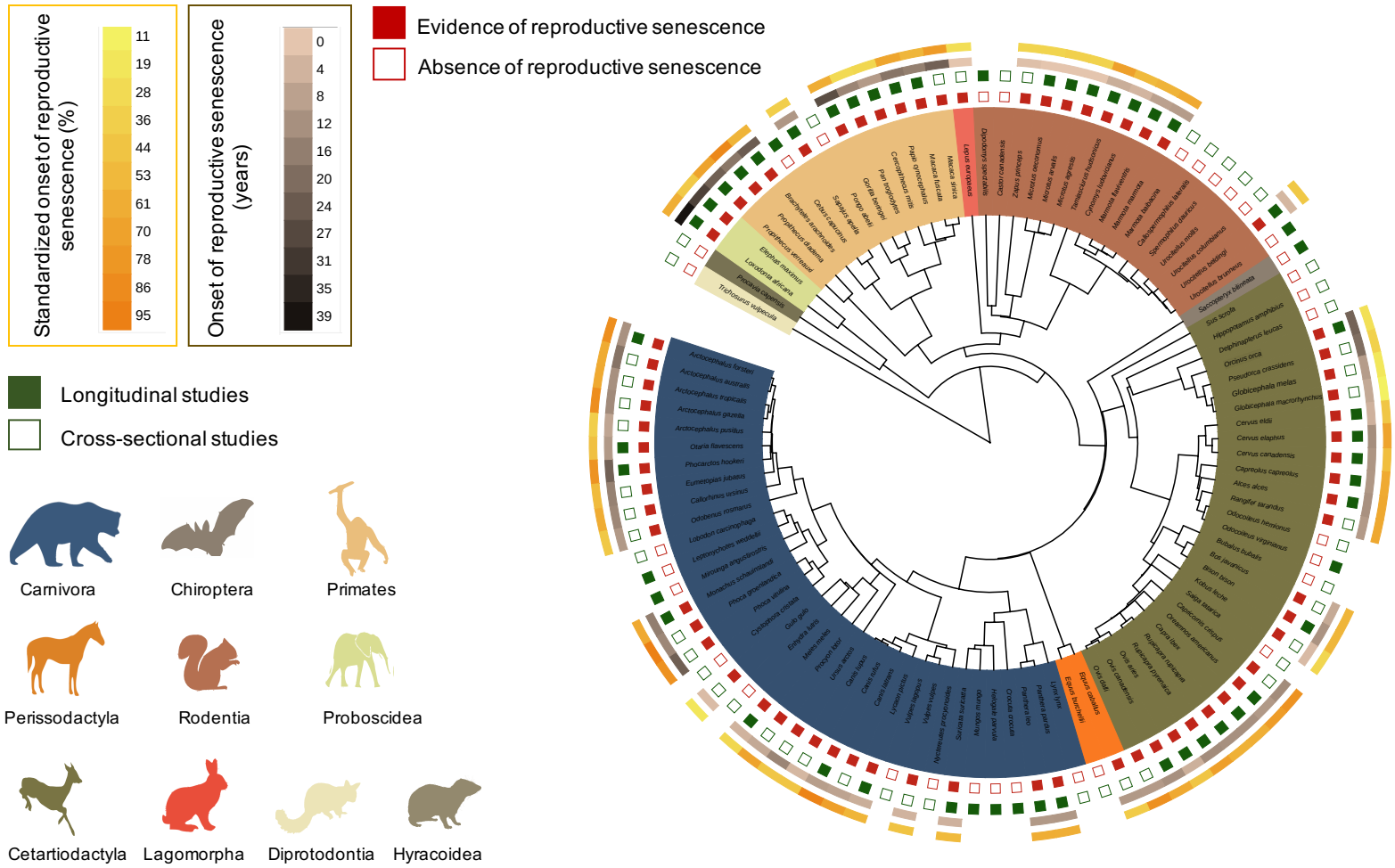

Standardized onset of reproductive senescence (%)
11
19
28
36
44
53
61
70
78
86
95
Onset of reproductive senescence (years)
0
4
8
12
16
20
24
27
31
35
39
Evidence of reproductive senescence
Absence of reproductive senescence
Longitudinal studies
Cross-sectional studies
Carnivora
Chiroptera
Primates
Perissodactyla
Rodentia
Proboscidea
Cetartiodactyla
Lagomorpha
Diprotodontia
Hyracoidea

FIGURE 1

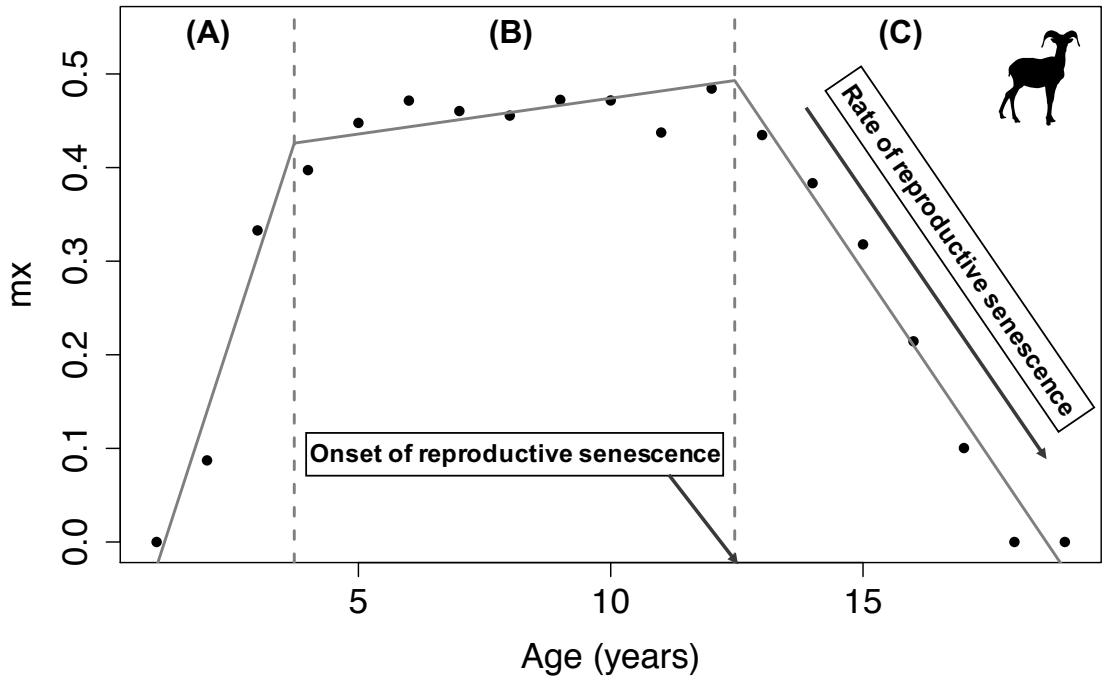

(A)
(B)
(C)
mx
0.0
0.1
0.2
0.3
0.4
0.5
5
10
15
Age (years)
Onset of reproductive senescence
Rate of reproductive senescence

FIGURE 2

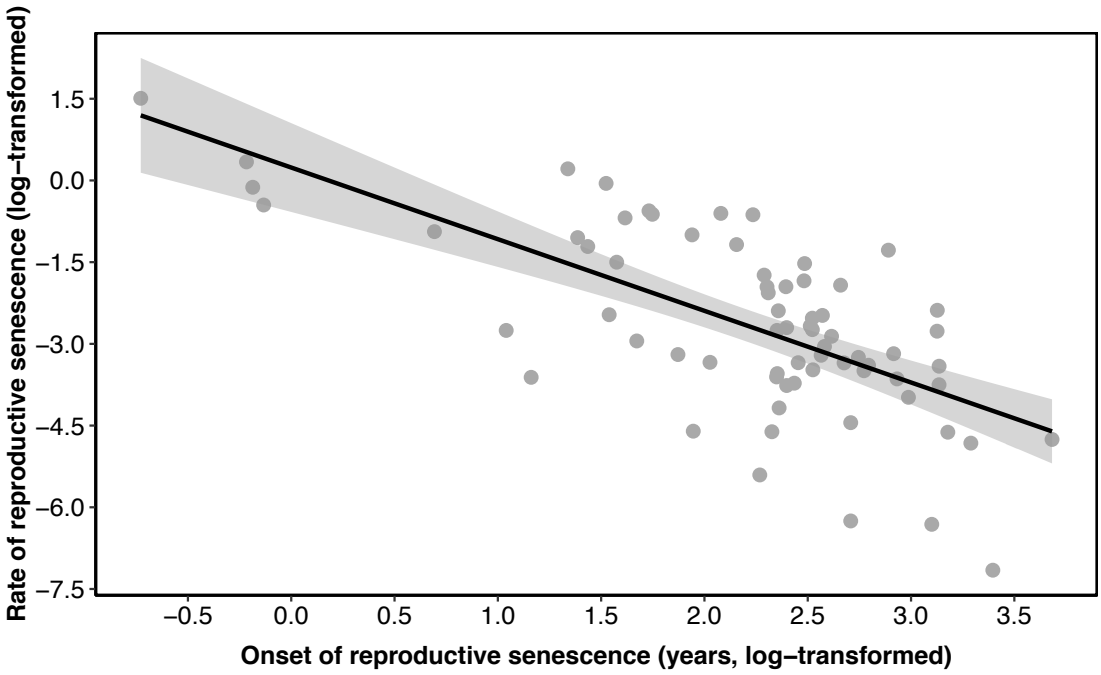

Rate of reproductive senescence (log−transformed)
1.5
0.0
−1.5
−3.0
−4.5
−6.0
−7.5
−0.5
0.0
0.5
1.0
1.5
2.0
2.5
3.0
3.5
Onset of reproductive senescence (years, log−transformed)

FIGURE 3

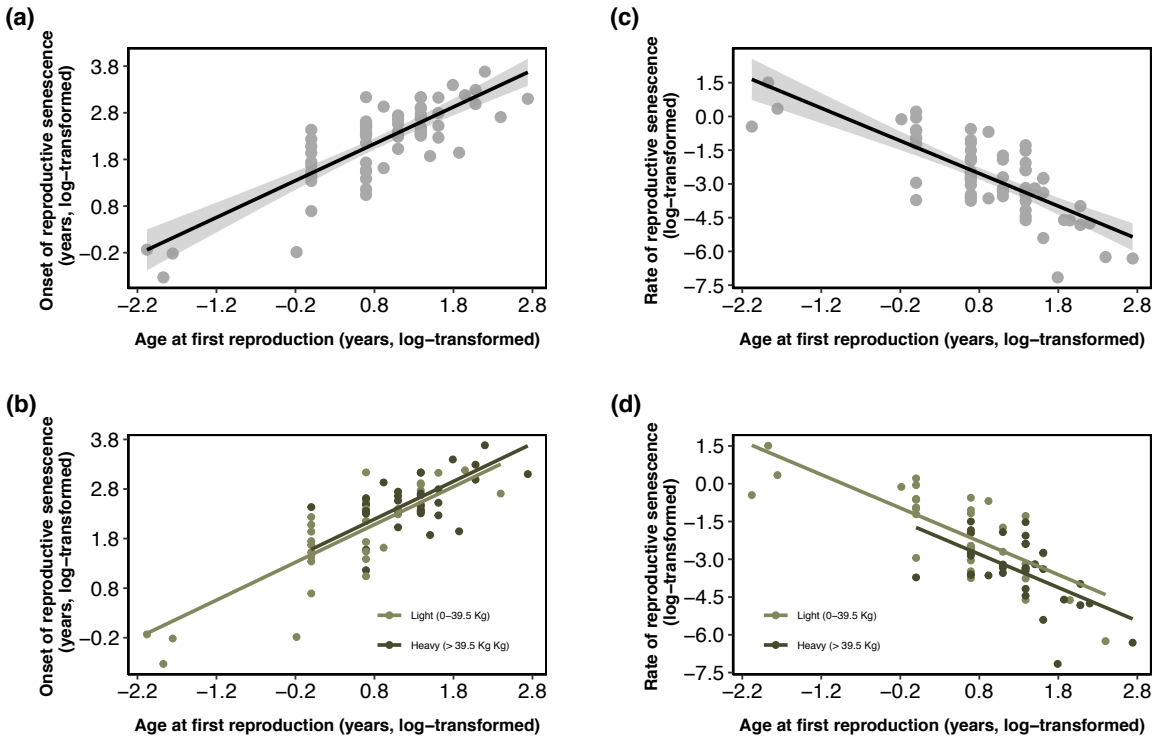
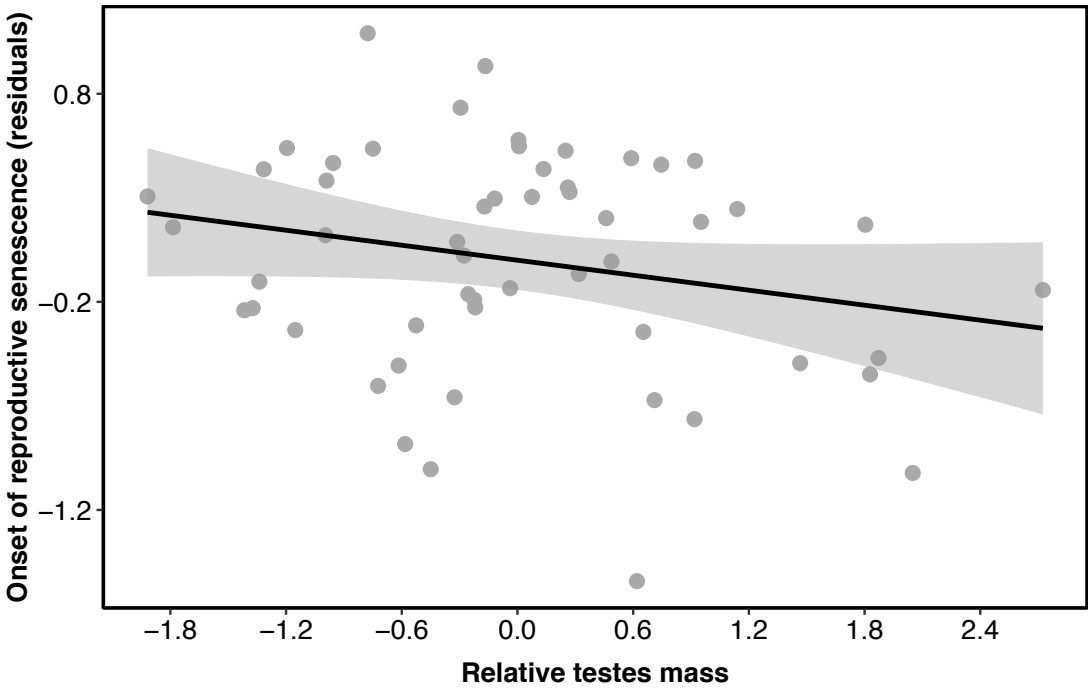
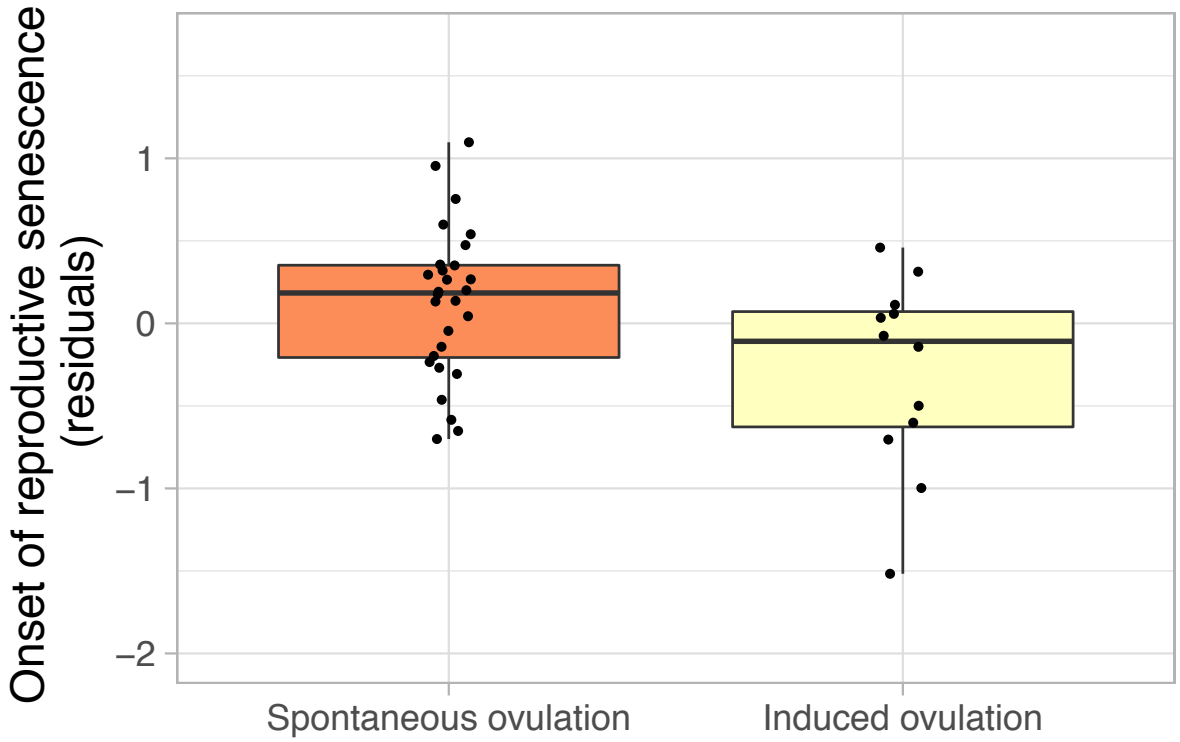

(a)
Onset of reproductive senescence (years, log−transformed)
3.8
2.8
1.8
0.8
−0.2
−2.2
−1.2
−0.2
0.8
1.8
2.8
Age at first reproduction (years, log−transformed)
(c)
Rate of reproductive senescence (log−transformed)
1.5
0.0
−1.5
−3.0
−4.5
−6.0
−7.5
Age at first reproduction (years, log−transformed)
(b)
Light (0−39.5 Kg)
Heavy (> 39.5 Kg Kg)
(d)
Light (0−39.5 Kg)
Heavy (> 39.5 Kg)

FIGURE 4



1071
1072
1073 **FIGURE 5**
1074



1075

1076 **FIGURE 6**